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BRITISH JOURNAL OF ORAL & MAXILLOFACIAL SURGERY

Fibrous dysplasia of the craniomaxillofacial region: current clinical perspectives

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SUMMARY. **Fibrous dysplasia is a benign fibro-osseous disease of bone of unknown etiology. Its occurrence in the craniomaxillofacial skeleton is frequent and varies in severity from an asymptomatic monostotic lesion to polyostotic involvement resulting in progressive functional deficit and aesthetic problems.**

With the advent of refined instrumentation and craniofacial surgical techniques, a more aggressive, non-disabling approach to these benign yet deforming fibro-osseous growths is possible. In some patients, complete excision of the involved bone with graft reconstruction of the resultant defect with primary autogenous bone may be possible. Lifelong continuous ongoing monitoring of the involved region is required throughout the patient's life.

Fibrous dysplasia is a benign fibro-osseous bone disease of unknown aetiology.[1,2] The range of skeletal involvement varies from an asymptomatic monostotic lesion to polyostotic involvement resulting in progressive functional deficit and aesthetic problems.[3–39] As part of the fibro-osseous process normal bone is replaced by cellular fibrous tissue and immature bone.[12,26] The clinical behaviour of fibro-osseous lesions varies greatly, though distinguishing histological features are few.[40]

Key points in the management of a patient with fibrous dysplasia are: the accuracy of the diagnosis; radiographic assessment; clinical evaluation; planning of interventions; procedures carried out; and the long term follow-up. Accurate diagnosis is important to understand better the natural history of the disease and the associated conditions including Albright's syndrome. The radiographic assessment should confirm the diagnosis and allow for assessment of the extent of skeletal involvement. A complete clinical evaluation will record any alteration of breathing, swallowing, speech, occlusion, vision, hearing and cerebral function. Thoughtful planning of the timing and extent of the intervention followed by precise operations when indicated will maximize both function and aesthetics. Lifelong continuing reassessment to monitor changes in the patient's needs throughout the patient's life is required.

HISTORICAL PERSPECTIVE

Fibrous dysplasia is a congenital dysplastic disease of bone that may occur in a single bone (monostotic) or many bones (polyostotic) and it is considered to be a type of fibro-osseous lesion.[3,10] Fibrous dysplasia of bone was first described by Von Recklinghausen in 1891 when he incorrectly included two cases of polyostotic fibrous dysplasia with his description of osteitis fibrosa cystica of hyperparathyroidism.[41] In 1937, Albright described a syndrome of polyostotic fibrous dysplasia that included: skeletal changes, cutaneous pigmentation, and endocrine disturbances (precocious puberty in girls being the most striking example).[42] In 1938, Lichtenstein reviewed the publications to date, further delineated the clinical spectrum, and clarified the pathological anatomy of this condition which he named fibrous dysplasia.

Aetiology and pathophysiology

The cause of the abnormal fibro-osseous process has not been clarified.[43–47] Macroscopically, the lesion looks yellowish or grey and is highly vascular.[26,44] Histopathological analysis shows that it consists of areas of fibrous tissue interwoven with newly formed bone. The fibrous tissue may be extensive or limited and may vary within areas of the same lesion. The bony trabeculae vary in shape. Some are slender and C-shaped (called 'Chinese' characters trabecula). There is no lamellar bone formation in the classic histological picture of the disease.[40]

Why there is maturational arrest at the woven bone stage is open to question.[40] Several authorities have suggested that the lesion is the result of trauma.[43,48] Lichtenstein indicated that it is caused by aberrant activity in the bone-forming mesenchymal tissue.[3] Most authorities consider it to be a non-neoplastic developmental lesion of bone.

Clinical and radiographic presentation

The process may involve a single bone, more than one bone in a single region (sphenoid wing region) or be disseminated throughout the body,[49] but the monostotic form of the disease is the most common.[5,14] Polyostotic fibrous dysplasia associated with precocious puberty and café-au-lait spots is known as Albright's syndrome.[42,50–51] Although both forms of the disease usually begin in childhood, age of onset and symptoms vary considerably from patient to patient and they usually seek medical attention once they are aware of symptoms or signs. Some are identified only on routine radiographic or physical examination. Harris *et al.* reported a patient with no symptoms whose disease was not diagnosed until he was 68 years of age.[48] At the time of diagnosis, the disease had affected 30% of the skeleton.

In many patients, the lesions are recognized in early childhood, grow slowly, and stabilize in early adult life. The point at which growth is arrested is unpredictable and is not always reached by puberty. Such lesions rarely degenerate into osteosarcomas,[52–57] which are more likely to follow a course of radiotherapy undertaken in an attempt to control the lesion. Radiation is of no value in the treatment of this condition, and the possibility of developing a late post-irradiation sarcoma of bone makes it an absolute contraindication.

The most commonly involved bones of the craniofacial skeleton are the maxilla and frontal bones. When the maxilla is affected, other adjacent bones separated by sutures (zygoma, sphenoid, frontal, nasal) are often also involved and so it is strictly not monostotic. When the frontal bone is affected, involvement of the adjacent sphenoid, temporal, and zygomatic bones is common.

The most usual finding is painless enlargement of the involved bone, which presents as facial asymmetry or a pathological fracture of an extremity.[12,22,29,58–59] The onset is often so gradual that the patient cannot remember when the swelling began. Leads and Seaman described 46 patients with fibrous dysplasia of the skull among whom a painless mass accounted for 45% of the initial symptoms and presentation.[44] Twenty-four percent were detected incidentally by radiography. Symptoms are more common when enlargement of the bones at the base of the skull cause narrowing of the neural foramina leading to blindness, tinnitus and deafness.[23,28,46,60–61] Proliferation of the tumour may also obstruct ostia and produce epiphora in the case of the nasolacrimal apparatus or sinusitis if the nasal ostia are involved. Partial obstruction of the nasal passages may result in altered resonance of speech, diminished nasal airflow with mouth breathing, and sleep apnoea.[59]

Fibrous dysplasia may affect the anterior base of the skull with inferior displacement of the orbital roof which results in proptosis of the eye.[29,32,39,60 63] If the maxilla is primarily involved with extensions to the orbital floor, the globe will be displaced upwards resulting in orbital dystopia. In either case, the lateral wing of the sphenoid bone and the optic canal may be narrowed, resulting in direct compression of the optic nerve(s) with subsequent papilloedema, optic atrophy, and blindness.

Differential diagnosis

Cherubism is also a fibro-osseous lesion with several features that distinguish it from fibrous dysplasia of bone. Cherubism is a benign, hereditary (autosomal dominant pattern) giant cell lesion of the jaws that presents in children as a bilateral painless swelling, generally between the ages of 2 and 5 years, with progression of the lesion until puberty when it often regresses spontaneously.[70–74] It generally requires little or no treatment but may displace teeth, distort facial features, and result in pathological fractures. Other developmental disorders of bone often confused with fibrous dysplasia include giant cell granuloma, aneurysmal bone cyst, and florid fibrous dysplasia. Ossifying fibroma is a true bone tumour and should be distinguished from these other lesions.[73 74]

TREATMENT

Radiotherapy is contraindicated because of its potential for malignant transformation and chemotherapy is not effective, so surgical intervention is the only treatment option. The timing and extent of operation as opposed to conservative observation requires clinical judgement and informed consent.[32–33,39,48,58–59,62–69]

Case reports

Five patients show the range of presentations and treatments that lend perspective to the clinical management of fibrous dysplasia of the craniomaxillofacial regions. (Figs 1–5).

CONCLUSIONS

With the advent of refined instrumentation and craniofacial surgical techniques, a more aggressive, non-disabling approach to those benign yet deforming fibro-osseous growths is possible. In some patients, complete excision of the involved bone with reconstruction of the resultant defect with primary autogenous bone may be possible. For others, despite the unpredictable nature of the residual disease, effective debulking of the regional fibrous dysplasia may be done with improvements in function, facial appearance, and self esteem. Lifelong continuous monitoring is required.

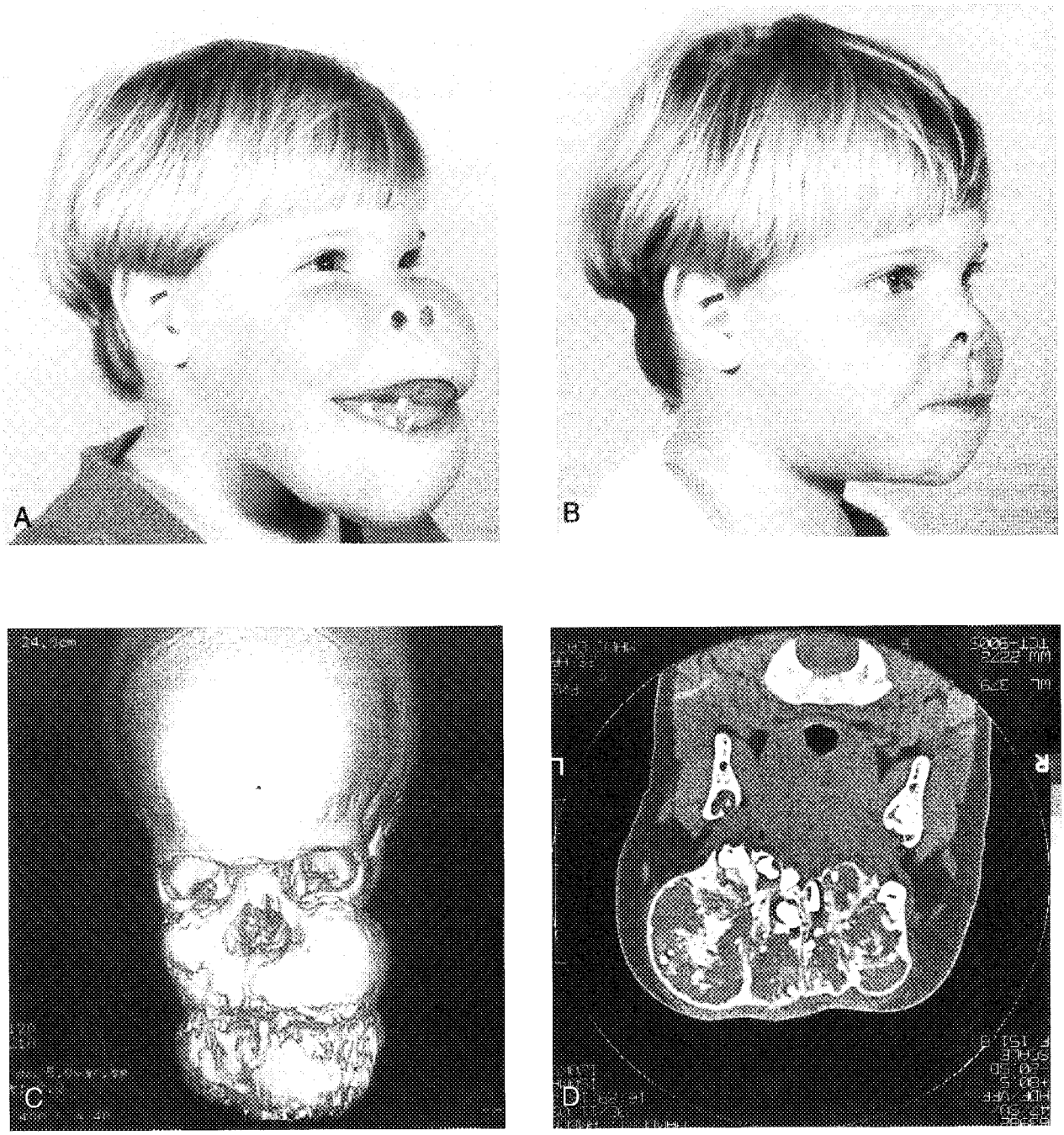


Fig. 1 A 5-year-old girl with polyostotic fibrous dysplasia and massive involvement of the maxilla and mandible bilaterally. An asymmetric swelling of the mandible (left more than right) was first recognized when the child was 18 months old: it rapidly enlarged and maxillary involvement became evident with deterioration of breathing, chewing, speech and swallowing. Pathological fractures were sustained in all four extremities as a result of involvement in fibrous dysplasia. (A) Preoperative oblique view. (B) Oblique view 10 days after undergoing radical maxillary and mandibular debulking procedure. (C) Frontal three-dimensional craniofacial CT view before operation. (D) Two-dimensional CT view through body and symphyseal region of mandible before operation. (From: Posnick JC, Hughes CA, Milmoe G et al: Polyostotic fibrous dysplasia: An unusual presentation in childhood. J Oral Maxillofac Surg. Reproduced with permission).

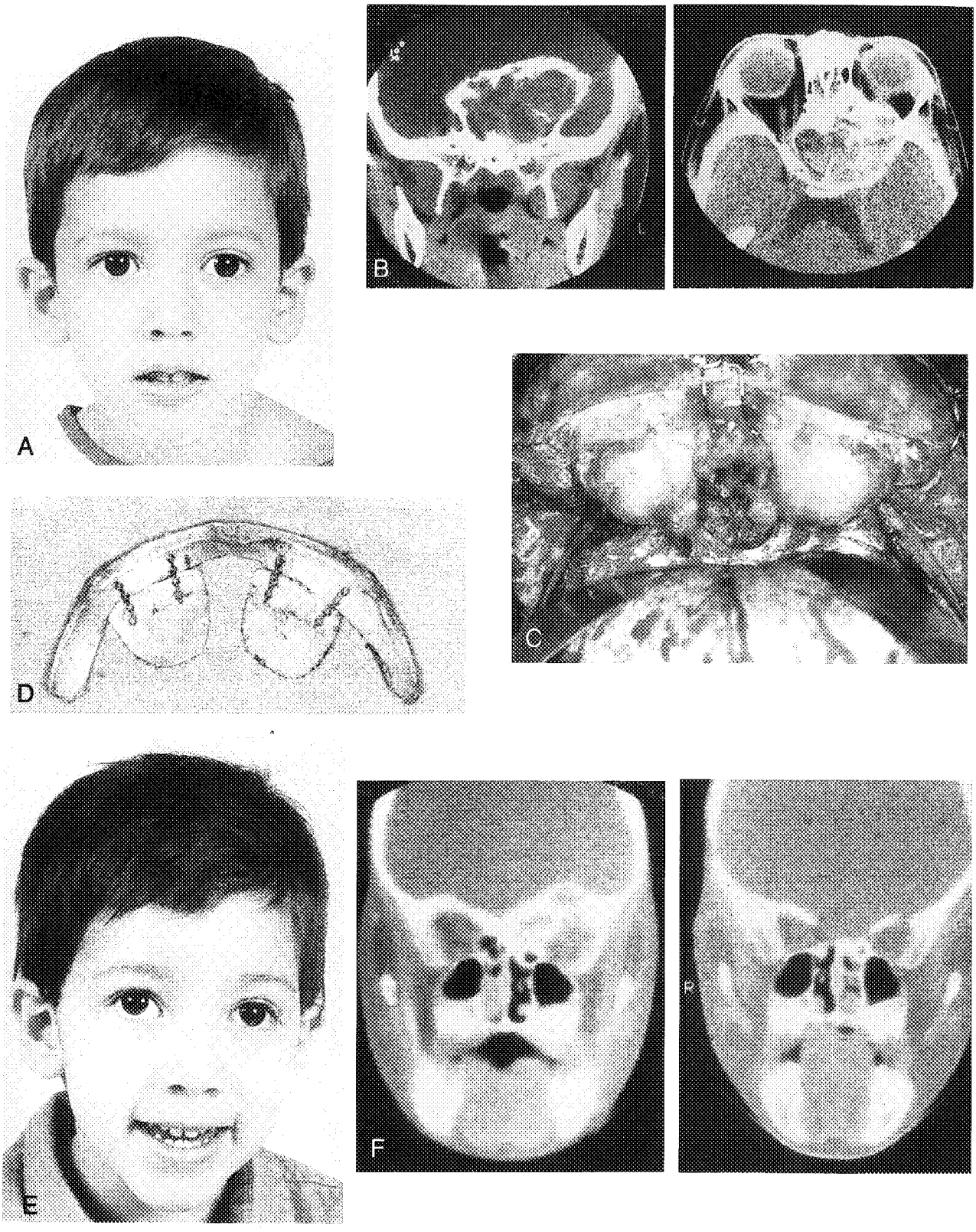


Fig. 2 – A 3-year-old boy presented to the emergency department with progressive visual loss and a degree of proptosis of the left eye. Both optic nerves were severely atrophic on fundoscopy, particularly in the left proptotic eye. A craniofacial CT and magnetic resonance imaging confirmed the diagnosis of fibrous dysplasia and showed the extent of involvement of the anterior base of the skull and orbits. (A) Frontal view at the time of presentation. (B) Coronal (*left*) and axial (*right*) CT slices through the cranial base and orbits, showing the extent of abnormal fibro-osseous bone resulting in compression of the optic nerve. (C) Close up view of optic chiasm after debulking of tumour. (D) The orbital roofs are reconstructed with autogenous split cranial grafts and fixed in place with microplates and screws to the upper orbital unit before being reinset. (E) Full-face view 1½ years after operation. (F) Coronal CT close to optic foramen before (*left*) and after (*right*) tumour resection and reconstruction of orbital roofs. (From: Posnick JC, Wells MD, Drake JM et al: Childhood fibrous dysplasia presenting as blindness: A skull base approach for resection and immediate reconstruction. Ped Neurosurg 1993; 19: 260–266. Reproduced with permission)

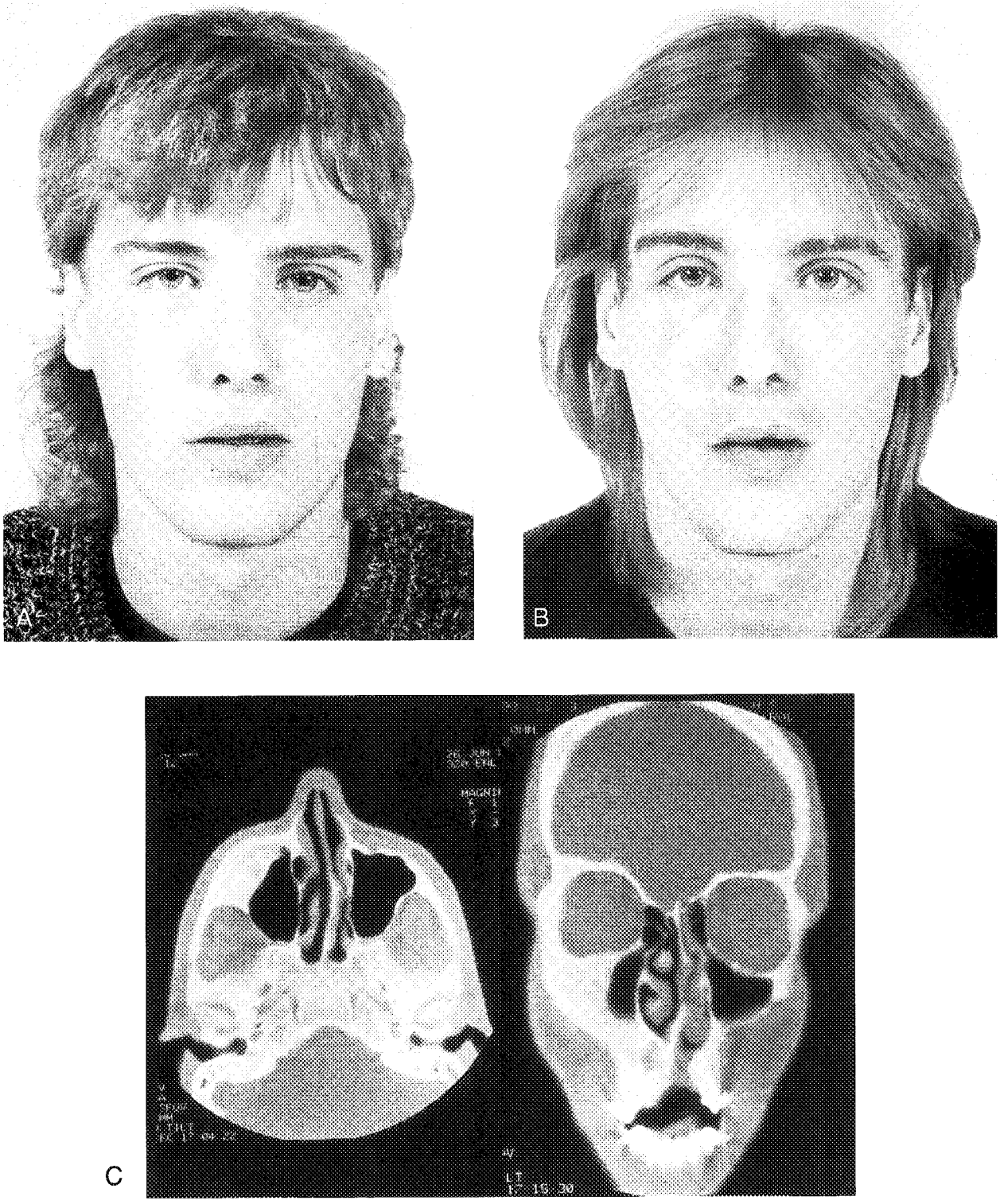


Fig. 3 – A 17-year-old boy born with an incomplete cleft of the left lip and palate. He underwent orthodontic treatment in preparation for planned orthognathic surgery and bone grafting of his alveolar defect. One month before surgery, he complained of diplopia of the right eye. Radiographs showed fibrous dysplasia of the right maxilla, orbit, zygoma and towards the base of the skull along the orbital floor. He underwent removal of fibrous dysplastic bone of the upper maxilla, anterior zygoma, lateral orbital and infraorbital rim, lateral orbital wall and floor with decompression of the orbital contents. Reconstruction comprised a pedicled temporalis muscle flap used to close the dead space of the maxillary sinus and then full-thickness autogenous cranial bone graft to reconstruct the anterior maxilla, anterior zygoma, lateral and infraorbital rims and lateral orbital wall and floor. (A) Full-face view before operation. (B) Full-face view 1 year after resection and reconstruction. (C) Coronal and axial CT slices through midface and orbit showing extent of fibrous dysplasia.

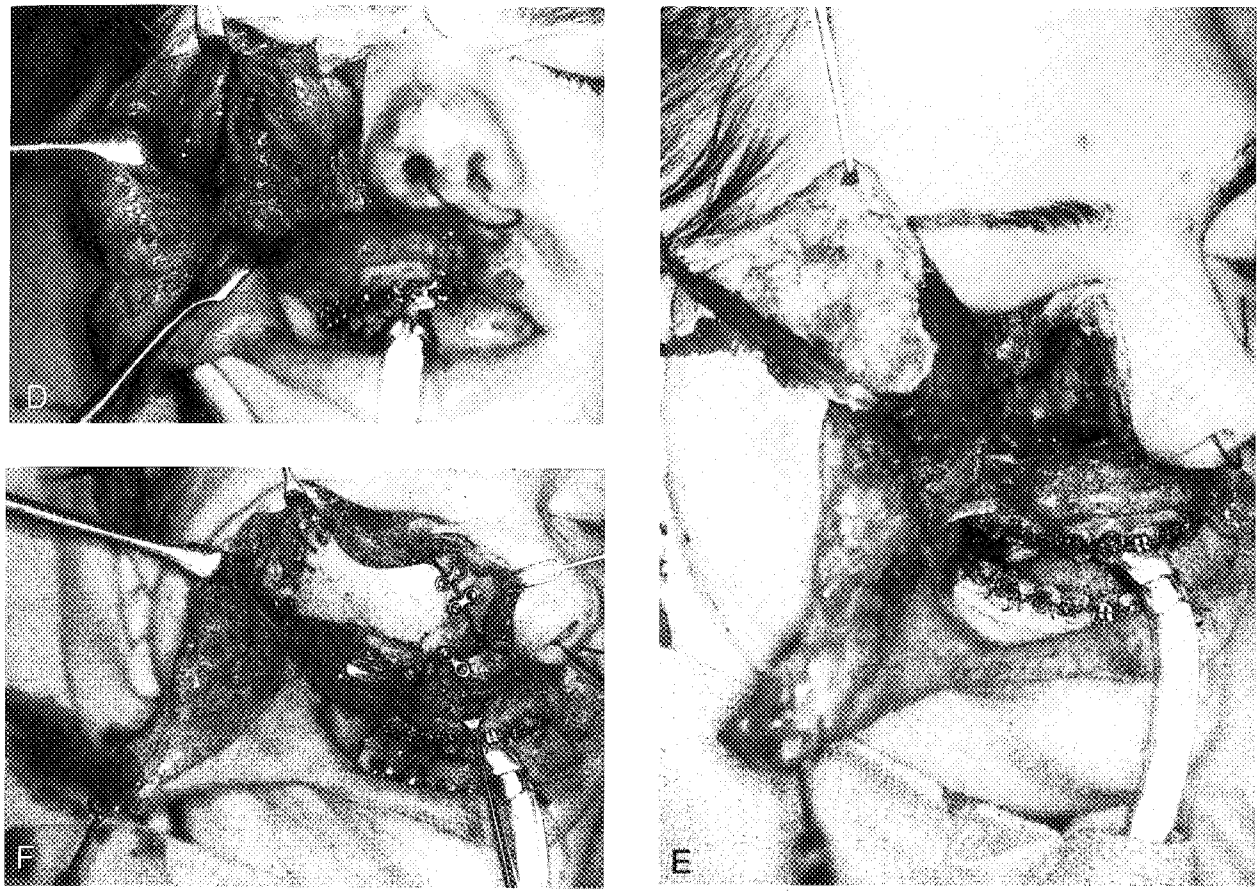


Fig. 3 – *(cont.)* (D) The orbital contents are decompressed by removal of abnormal fibrous dysplastic bone. (E) Elevation of anterior half of temporalis muscle through coronal incision. (F) View of temporalis muscle flap obliterating dead space of maxillary sinus with overlying reconstruction of skeletal defect with cranial bone.

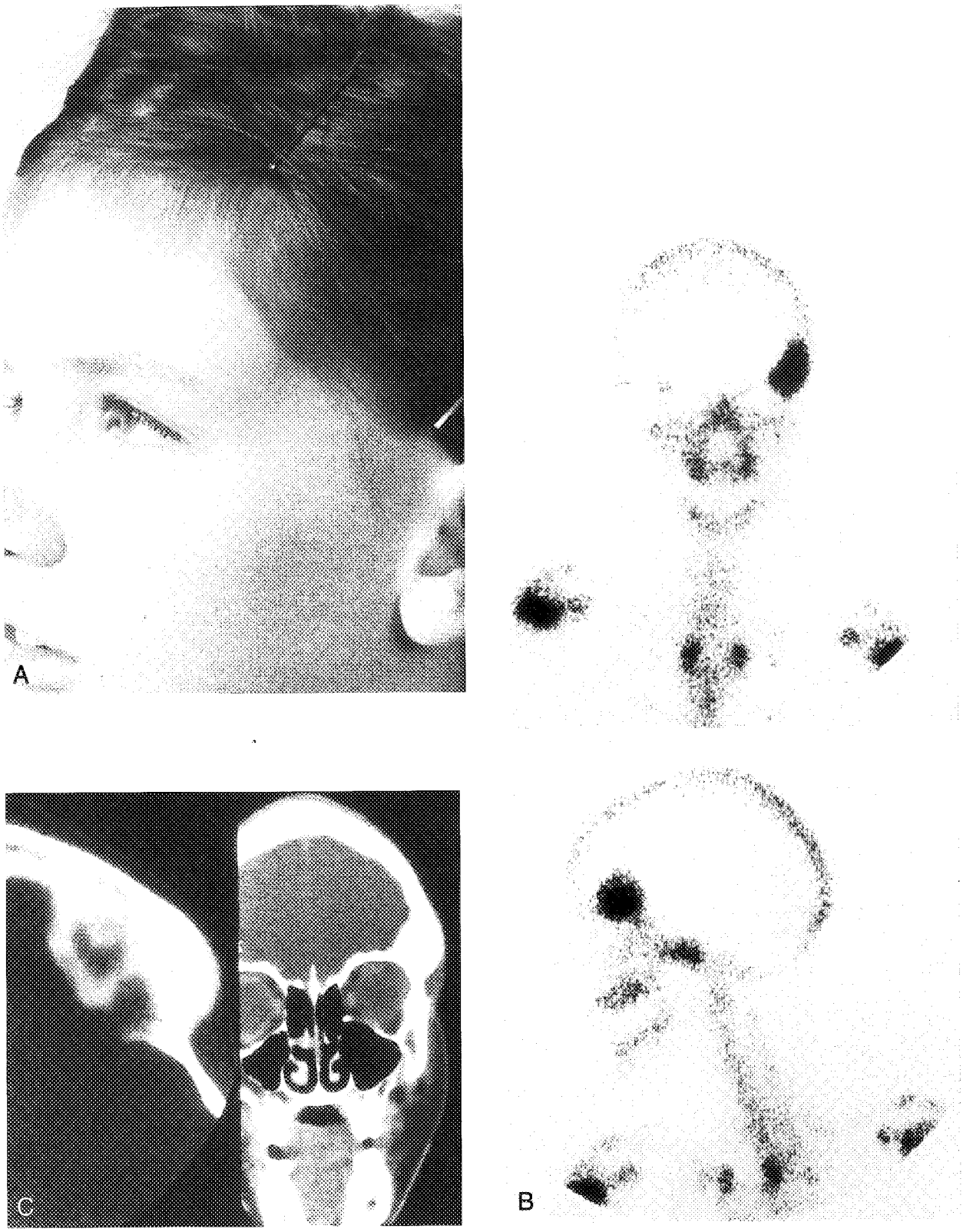
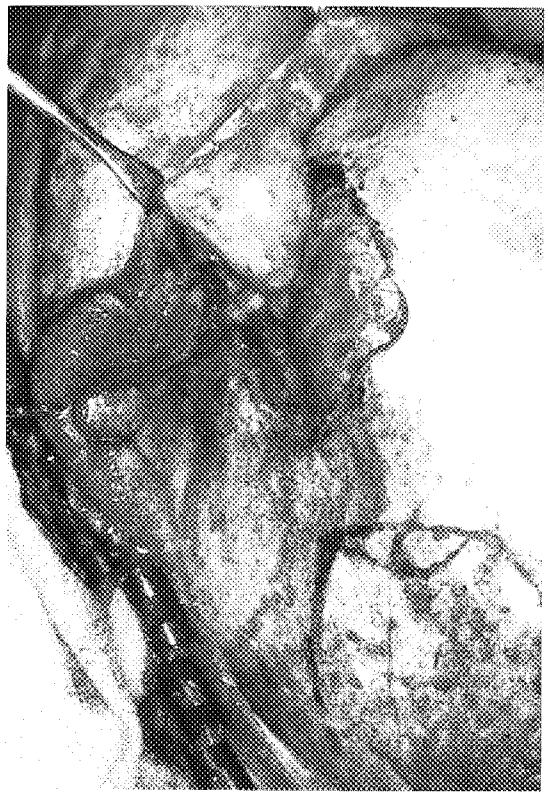
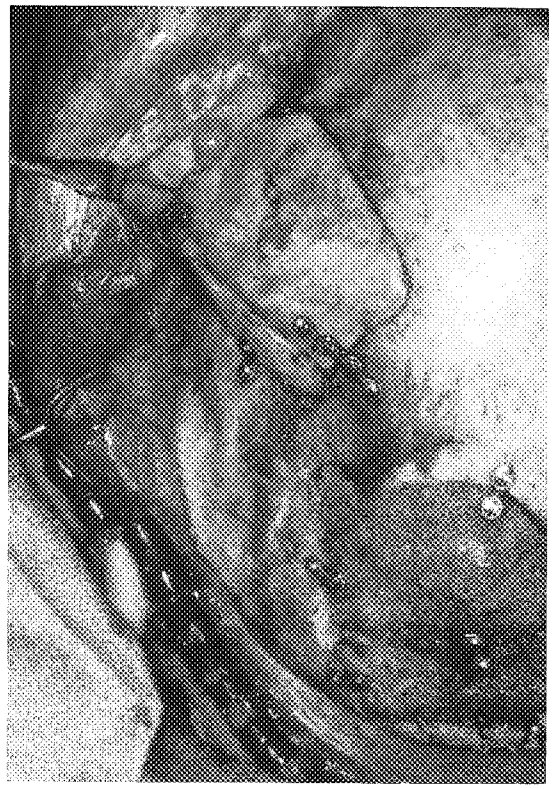
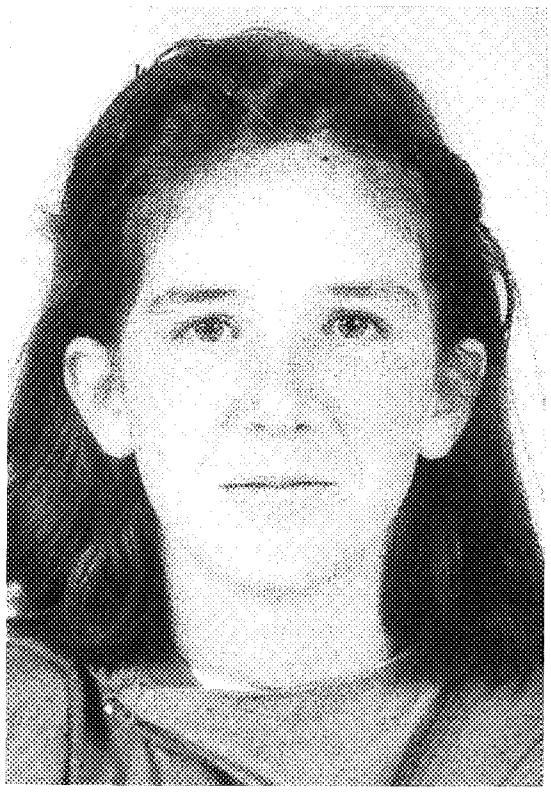


Fig. 4 – An 8-year-old girl had a left fronto-orbital and sphenoid wing monostotic fibrous dysplasia which presented as a painless, hard mass, followed by diplopia and headache. An ophthalmological assessment and CT scan confirmed the extent of involvement. (A) Close-up facial view showing left frontotemporal mass. (B) Bone scan confirms the area of reactivity of the left fronto-orbital and sphenoid wing region. (C) View through the coronal incision showing the mass in the left fronto-orbital and sphenoid wing mass.







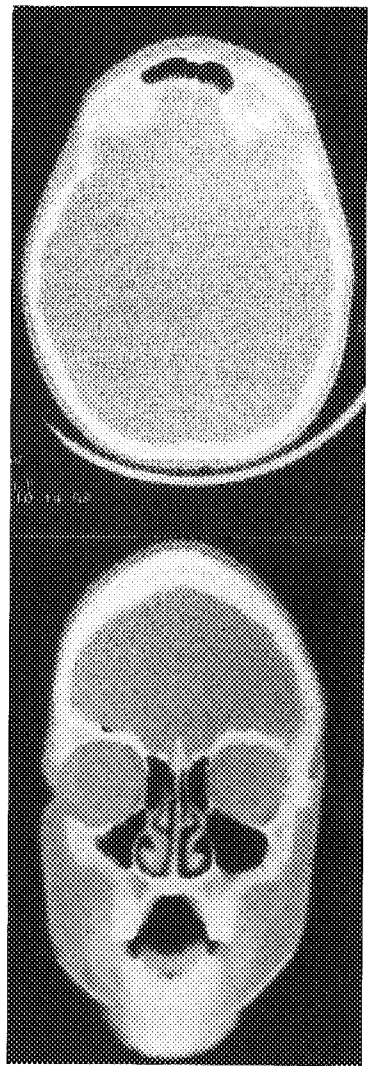


Fig. 4 – *(cont.)* (D) The tumour mass is resected back to the optic foramen. (E) Cranial bone graft reconstruction. Stabilization is achieved with multiple titanium bone plates and screws. (F) Frontal view shows the patient 4 years after resection and reconstruction. (G) Two-dimensional CT scan views through orbit demonstrate reconstructed region. (From: Posnick JC: The role of plate and screw fixation in the management of pediatric head and neck tumours. In: Gruss JS, Manson PM, Yaremchuk MJ, eds. Rigid fixation of the craniomaxillofacial skeleton. Stoneham, MA, Butterworths, 1992: 665. Reproduced with permission).

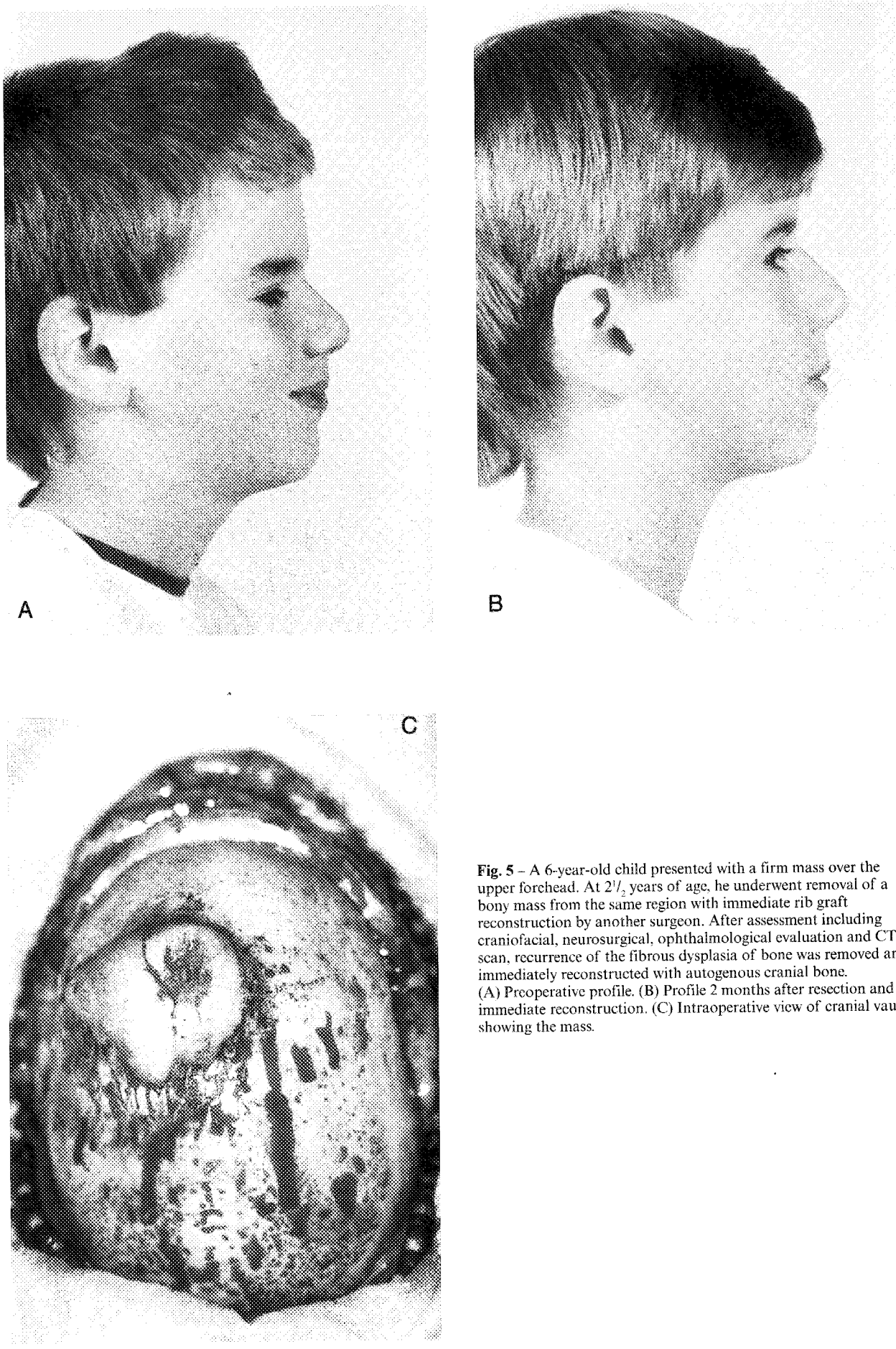


Fig. 5 – A 6-year-old child presented with a firm mass over the upper forehead. At $2^1/_2$ years of age, he underwent removal of a bony mass from the same region with immediate rib graft reconstruction by another surgeon. After assessment including craniofacial, neurosurgical, ophthalmological evaluation and CT scan, recurrence of the fibrous dysplasia of bone was removed and immediately reconstructed with autogenous cranial bone. (A) Preoperative profile. (B) Profile 2 months after resection and immediate reconstruction. (C) Intraoperative view of cranial vault showing the mass.

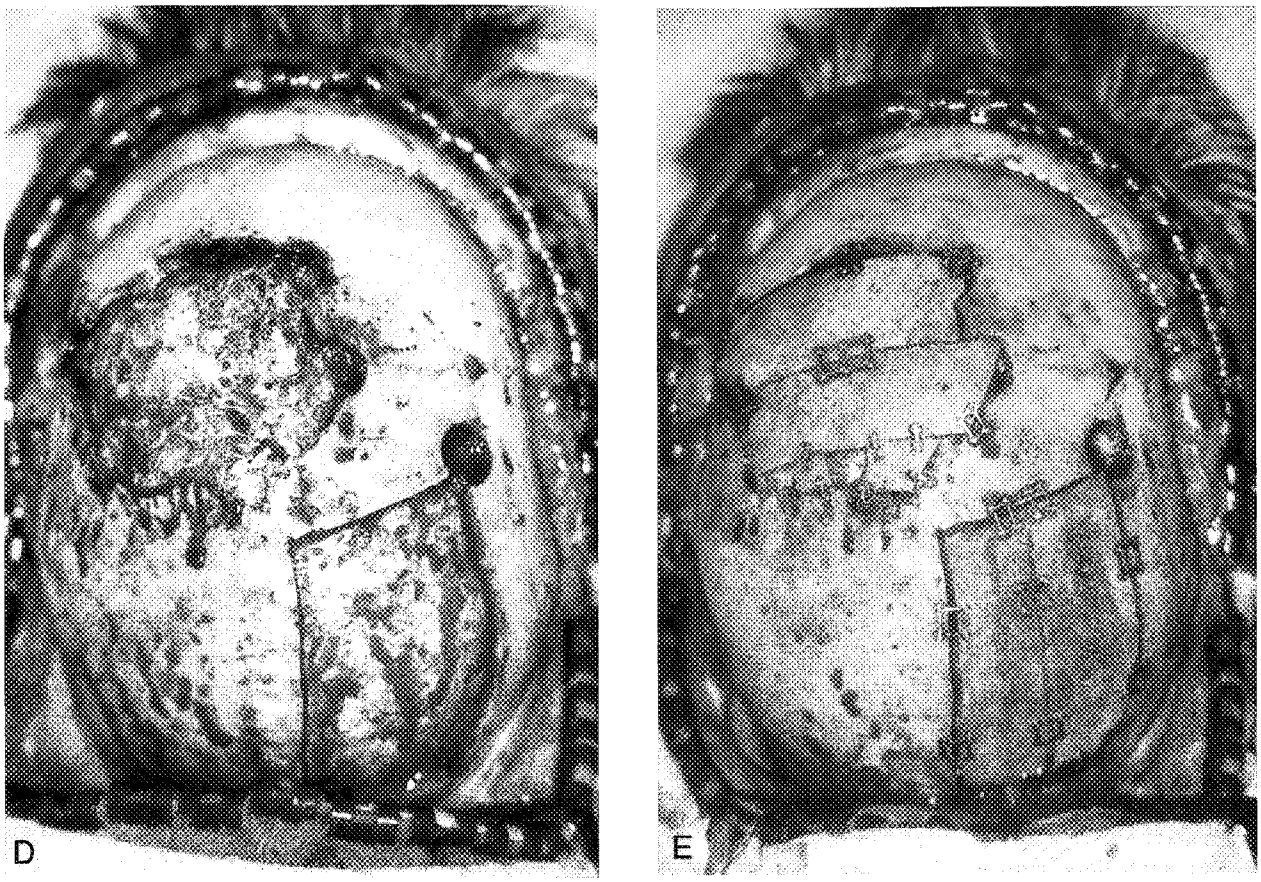


Fig. 5 – *(cont.)* (D) Intraoperative view after removal of the left frontotemporal mass and harvesting of full-thickness right occipitoparietal cranial bone for reconstruction. (E) View of cranial vault with donor (frontotemporal) and recipient (occipitoparietal) sites reconstructed with split autogenous cranial grafts fixed with microplates and screws.

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